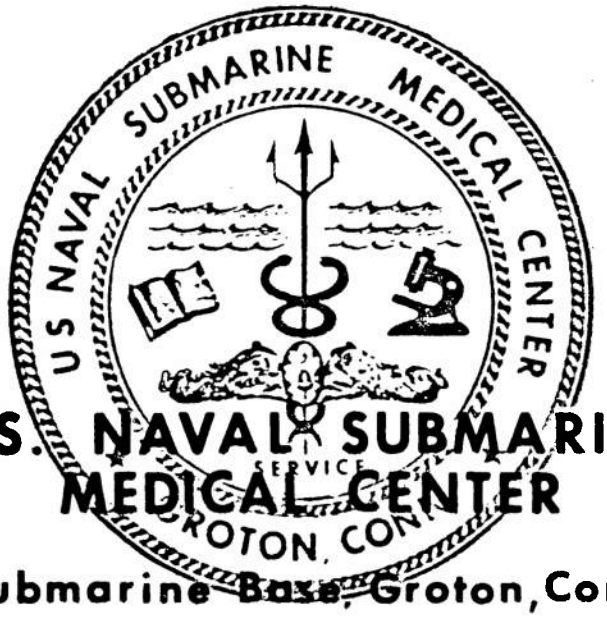

U. S. NAVAL SUBMARINE MEDICAL CENTER
Submarine Base, Groton, Conn.

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CASUALTIES IN INDIVIDUAL SUBMARINE ESCAPE

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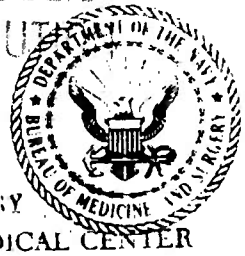

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CASUALTIES IN INDIVIDUAL SUBMARINE ESCAPE TRAINING

OUTLINE

I. Historical Review of Individual Submarine Escape

- A. S.E.A.
- B. Free Ascent
- C. Buoyant Ascent
- D. Limitations of scope of this paper

II Summary of Casualties

- A. Introduction
  - 1. Source of data
  - 2. Location of training casualties
- B. Clinical factors in the etiology of submarine escape training casualties
  - 1. Review of training procedures
  - 2. Etiological factors in S.E.A. casualties
  - 3. Etiological factors in free ascent casualties
  - 4. Etiological factors in buoyant ascent casualties
    - a. Techniques at New London and Pearl Harbor
  - 5. Entrapped air spaces
  - 6. Etiological factors in recompression chamber fatalities
- C. Clinical characteristics of submarine escape training casualties
  - 1. Air embolism
    - a. Review of etiological factors
    - b. Ascents
      - (1) Past
      - (2) Day of ascents - depth and number
      - (3) Depth of accident
        - (a) Survivors
        - (b) Fatalities, including methods of ascent
    - c. Onset of signs and symptoms - depths and/or time
      - (1) Survivors
      - (2) Fatalities

d. Symptoms and signs

(1) Listing, with number of cases
    (a) Survivors and fatalities
    (b) Fatalities

e. Interval between onset of symptoms and signs and beginning of treatment

(1) Survivors
(2) Fatalities

f. Treatments

(1) Review of history
(2) Survivors
    (a) Recurrences - (see extra-alveolar air)

(3) Fatalities

g. Post-treatment examination

(1) X-rays of survivors
(2) Autopsies of fatalities

2. Extra-alveolar air

a. Listing

(1) Isolated Cases
(2) Cases occurring with air embolism

b. Ascents

(1) Past
(2) Day of accident - number and depth
    (a) Just prior to appearance of extra-alveolar air
    (b) Type of ascent
        1. Those occurring with air embolism
        2. Isolated extra-alveolar air

c. Onset of signs and sumptoms - depth and/or time

(1) Those in air embolism cases
(2) Isolated extra-alveolar air

d. Signs and symptoms

(1) Listing with number of cases
(2) Discussion

e. Time interval between onset of symptoms and signs and beginning of treatment

f. Treatment

(1) Those occurring with air embolism
(a) Recurrences

(2) Isolated extra-alveolar air

g. Post-treatment examination

(1) X-ray

3. Miscellaneous

a. Listing
b. Decompression illness

(1) Etiological factors
(2) Onset of symptoms and signs
(3) Symptoms and signs
(4) Treatment

c. Vestibular injury
d. Pulmonary squeeze
e. Hysterical syncope

(1) Confusion with air embolism

D. Clinical characteristics of recompression chamber casualties

1. Introduction and etiological factors
2. Air embolism

a. Preceding exposures to pressure
b. Onset of signs and symptoms - depth and/or time
c. Signs and symptoms - with number of cases

(1) Differential diagnosis

d. Interval between onset of signs and symptoms and treatment

e. Treatment

(1) Recurrences

f. Post-treatment examination (X-rays)

3. Extra-alveolar air

a. Onset

(1) Those occurring with air embolism
(2) Isolated extra-alveolar air

b. Signs and symptoms - listing with number of cases
c. Treatment

(1) Interval until begun and actual treatment

(a) In air embolism cases
(b) Isolated extra-alveolar air

d. Post-treatment examination (X-rays)

4. Miscellaneous

a. Decompression illness
b. Hysteria
c. Etc.?
d. Allergy ?

E. Statistical analysis of escape training casualties

1. Introduction
2. Incidence of casualties at all three tanks

a. Aeroembolism

(1) Casualties/total number of ascents
(2) Fatalities/total ascents
(3) Fatalities/total casualties

b. Extra alveolar air

(1) Casualties/total ascents

3. Incidence of water training casualties by year and method of ascent

a. Comparison with New London tank workload (as representative)

4. Chamber casualties

5. Escape training experience at the New London tank

a. Introduction
b. Combined incidence of air embolism and extra-alveolar air casualties for different methods of escape

F. Summary

G. Appendix - Breakdown of all recorded cases

I. Brief History of Individual Submarine Escape Methods.

A. S.E.A.

As a result of the large loss of life resulting from the sinking of its submarines in the second and third decades of this century, the U.S. Navy began investigating safe methods of individual escape from bottomed submarines. The project was initially directed by LT C. B. MOMSEN, who had designed his "lung" (or submarine escape apparatus). This was essentially a closed-circuit SCUBA, with a reservoir bag, a CO_2 absorbent-containing cannister, and a flutter valve to vent the expanding gas in the bag on ascent. The device was strapped to the chest, inflated with air or O_2, and could carry the survivor to the surface, subsequently being utilized as a life jacket. Experiments with, and limited use of this device began in the late 1920's, mainly at the mine tank at the Naval Gun Factory, and in the open sea from diving bells. The Escape Training Tanks at New London and Pearl Harbor were built to provide training for the fleet, the former commissioned in 1930, the latter in 1932. Initially, the New London tank was operated by the crew of the salvaged submarine hull S-4, which was also used for experiments on submarine escape techniques. They travelled to other facilities as well, training crews in submarine escape techniques. In the early days of the tanks, the operating personnel observed trainees through portholes in the sides, not entering the water except to operate a roving diving bell. Later, probably in the late 1930's, the instructors mastered skin-diving techniques enough to maintain close underwater supervision.

B. Free Ascent

In 1946, at the New London Tank (in 1951 at Pearl Harbor), instructors and select trainees began regularly using the technique of free ascent (or free escape), although it was used unofficially for many years previously. The method is based upon the principle that an individual, buoyant with total lung volume at the surface, will be buoyant at any depth with his lungs fully inflated. The object then is to maintain enough air in the lungs at a given depth to afford positive buoyancy, and yet not allow the pressure of the expanding intra-alveolar air to exceed 60-100mm Hg (3-4 ft. of water). Should the latter occur, the air will enter extra-alveolar tissues and blood vessels, with potentially grave results.

C. Buoyant Ascent

In 1946, the British Admiralty began investigating the known cases of escape from actually sunken submarines of all nations. It was found that, in those cases studies, most of the successful escapes

were made not with the use of a S.E.A. (Momsen Lung, British Davis S.E.A., German Draeger apparatus - all esentially similar), but rather by free ascent or buoyant-assisted free ascent (buoyant ascent). This was a result of malfunctioning S.E.A.'s and/or poorly-trained crews. The buoyant ascents were made using inflated S.E.A.'s, life jackets, pillowcases, etc. After preliminary investigations the British built their submarine escape training tank (at Gosport) to train their crews in buoyant ascent techniques. The U.S. Navy also investigated this technique, and it was adopted as its official method of individual submarine escape, to be taught to the entire fleet, in late 1956. Essentially, the method uses an inflated life jacket, as an assurance of positive buoyancy to carry the escapee to the surface.

D. Limitations of scope of this report

This report is intended only as a presentation of the three methods of individual submarine escape (S.E.A., free ascent and buoyant ascent) by an analysis of the reports of casualties and available statistics. It does not, however, exhaustively cover air embolism theory, collective and individual submarine escape theories and techniques, or techniques of flooding compartments preparatory to escape. It is rather a clinical evaluation of submarine escape training experience.

II. Summary of Casualties

A. Introduction

Available as clinical reports, or recorded on NAVMED 816's (U.S. Navy Diving Accident Reports) are 62 cases of casualties resulting from water work involved in submarine escape training, from 1928 to 1957. Of these, 39 were in the New London tank, 11 in the Pearl Harbor tank, 3 in the Mine tank at the Naval Gun Factory, 1 at NMRI, and 8 in the open sea. Seventeen (17) recompression chamber casualties were associated with submarine escape training, 11 at New London, 4 at Pearl Harbor, 1 on a tender (U.S.S. ORION), and 1 reported by the British tank at Gosport.

Numbered reference is made throughout this report to the breakdown of cases presented in the appendix.

B. Clinical factors in the etiology of submarine escape training casualties.

1. Review of training procedures

Training in individual escape from submarines has usually consisted of a pressure test to 50 p.s.i.g. (112 ft. - the maximum depth

of the escape training tanks) in a recompression chamber, and at least three escapes from various depths in the tanks. The latter varied somewhat since the inception of training, depending on the method(s) of escape in use and the workloads of the tanks. It has usually consisted of escapes from 18, 50 and possibly 100 ft. At times individuals with especial interest were allowed more than the minimal apparatus training, with additional instruction in free ascent. At the present time, instruction for the average U.S. Navy trainee consists of a 50 p.s.i.g. pressure test in the recompression chamber, preliminary "ladder training" to about 10 ft., one 18 ft. and two 50 ft. buoyant ascents in the water. Only instructors and SCUBA operators are taught free ascent and escapes from deeper than 50 ft.

2. Etiological factors in S.E.A. casualties

In theory, submarine escape using the S.E.A. should have no higher casualty rate than the average closed-circuit SCUBA rig. The device is a "circular" type of re-breathing apparatus, requiring only that the individual respire through the mouthpiece of a previously-charged bag. In some cases, the casualties were due to losing the mouthpiece, or failure to use the device properly. The most common cause for this was a phenomenon noted by many submarine medical officers: The trainee would complain of inability to respire into the device, commonly because of too much back-pressure in the bag, rarely due to a so-called "lack of air". This would usually occur at about 50' to 30' on the ascent, and would be accompanied by dizziness, or a feeling of suffocation, with resultant panic. The trainee would then let go of the ascending line and swim quickly to the surface, holding his breath or releasing it too slowly. The important feature here is that, essentially the individual was making a buoyant or fast free ascent on the upper portion of his ascent. S.E.A. ascents, correctly made, with adequate re-breathing into the bag, at a slow rate of ascent, do not seem to have resulted in any casualties (with the possible exception of some poorly-documented cases: Nos. 4, 5, 6, 7, and 8). All of the S.E.A. casualties seem to have occurred in the aforementioned manner.

3. Etiological factors in free ascent casualties

With the advent of free ascent training came the additional difficulty of attempting to maintain intra-alveolar pressure below a critical value by exhaling air, but, at the same time providing adequate buoyancy for ascent. Subjectively, the individual's only gauge for this fine balance was keeping "behind" or abreast of his bubbles. If he exhaled too much, he would have to swim a short distance to regain buoyancy. Those who rose faster than their bubbles (an empirical standard) or swam too high to regain buoyancy could possibly exceed the

critical intra-alveolar pressure. There are, however, apparently correctly performed ascents, to be discussed later, which do result in casualties.

4. Etiological factors in buoyant ascent casualties

There is a small percentage of individuals who naturally lack buoyancy with fully-expanded lungs (negative buoyant). Also, if a normally-buoyant individual attempts an excape in black or murky water, he may lose his orientation, not being able to determine whether he is rising or falling, and not being able to see his bubbles to gauge the speed of his ascent. Because of these two factors, plus the aforementioned finding that the majority of successful individual escapes from actually disabled submarines were free or buoyant ascents, buoyant ascent was chosen as the currently accepted method of individual submarine escape. Due to the very fast ascent with an inflated life jacket, transthoracic pressure increases rapidly. The escapee must maintain a margin of safety by keeping ahead of the pressure build-up. Failure to do this might result in air embolism or extra-alveolar air extravasation.

Initially, the Pearl Harbor and New London tanks differed in their techniques At New London, the escapee would hyperventilate in the escape air lock, fill his lungs to full capacity, begin exhaling a small quantity of air, and then exhale constantly all the way to the surface. At Pearl Harbor, however, the escapee would perform similarly, except that he would exhale a very large quantity of air almost down to residual volume before beginning his ascent, and then exhale any accumulated gas from the expanding lung volume during his entire ascent. At the present time all training tanks are using the latter method. Theoretically, the expansion of the residual volume in an average individual through an ascent of 2.5 atmospheres 50 ft. would not exceed the total lung capacity. So fat, in the escapee with normal lungs, this has proven to be the case

5. Entrapped air spaces

An important hazard of free ascents has been re-emphasized in buoyant ascent training. It is the possibility of entrapped air spaces in the lungs resulting from pre-existing lesions, congenital anomalies, or mucous plugs. Concentrated efforts at the autopsy table, as well as routine post-traumatic X-rays, have recently demonstrated lesions which seem to have effected a ball-valve action on air inspired under pressure. The air thus entrapped, on expansion offers a circumscribed area of excessive intra-alveolar pressure. This results in extra-alveolar extravasation, even though the individual is exhaling properly; obviously, it would make little difference here whether the

individual exhales completely before or during ascent - the air remains entrapped behind its barrier.

6. Etiological factors in recompression chamber casualties

Although differing in the environment in which they occur, recompression chamber casualties tend to follow somewhat the same pattern as those encountered in water training; since this is the trainee's first exposure to pressure in a small, closed compartment, it is utilized as a test of emotional stability as well as for the ability to equalize middle ear and sinus spaces with the rising ambient pressure. Frequently, instances of hysteria and anxiety reaction occur, indistinguishable from organic damage. As a result of their efforts to force air into their Eustachian tubes or sinus orifices, candidates may develop extremely high pharyngeal and transthoracic pressures. This, plus the possibility of entrapped air spaces, simulate the conditions of an improperly performed free or buoyant ascent. Traditionally, the pressure test has been to a maximum of 50 p.s.i.g. (112 ft.). Supposedly, if a trainee could equalize to this depth, he could at least be able to start any escape from the bottom of the tank.

C. Clinical characteristics of submarine escape training tank casualties

1. Air Embolism: Of the 62 escape training casualties directly or indirectly related to waterwork, 44 showed clinically and/or on autopsy the signs and symptoms of air embolism. Eight (8) of these were fatalities. Air embolism has many causes, their manifestations depending upon the site of entrance of gross air into the vascular system (venous or arterial).

The circumstances in submarine escape are those of any diving situation: The lungs are filled under increased atmospheric pressure, and, as the escapee rises through the water, the air within the lungs expands. The tissues of the lung have certain limits of elasticity, and when the transthoracic pressure becomes 60-100mm Hg greater than the ambient pressure, gross bubbles of air are found to extravasate into the extra-alveolar tissues and blood vessels. Should an individual vent off his lung air before the pressure builds up to the critical range, he can avoid this, assuming there is no entrapped air space.

These gross intravascular air bubbles, whatever their method of entry, are carried via the pulmonary vein to the left heart and into the aorta. The clinical signs and symptoms of air embolism are caused primarily by air bubbles carried to, and blocking cerebral vessels, and, in some cases, the coronary arteries. (Early attempts at treatment used a head-down position to lessen the former, with equivocal or negative results).

Thirty-one (31) cases of aeroembolism had undergone previous ascent training and/or pressure tests the same day; only 10 had performed escapes in the past. The number of ascents per man on the day of the casualty ranged from 1 to 5, averaging 3, their depths varying from 7-1/2 to 100 ft. Of the depth of ascent of the actual casualty, 8 ascended from 30 to 35 ft.; 5 from 12 to 18 ft.; 18 from 50 ft. and 13 from 100 ft. (some varying 10 ft). One death was from 15 ft., one from 30 ft., 3 from 50 ft. and 3 from 100 ft. ascents. Of these fatalities, the 15 and 30 ft. ascents were using S.E.A.'s, and were performed in the open sea from diving bells. Neither case regained consciousness, and there is no record as to whether the S.E.A.'s were functioning or used properly. Two (2) of the 100 ft. ascent fatalities were using S.E.A.'s and were struck before surfacing. One of these (No. 14) held his breath and the other (No. 21) lost his mouthpiece, so that, in effect, both made improper free or buoyant ascents holding their breath. Two (2) 50 ft. (Nos. 34 and 36) and 1 - 100 ft. (No. 45) ascent fatalities were free ascents; the former two were struck upon surfacing, the latter while ascending. All died before or immediately after being placed in the recompression chamber. They were thought to have made well-controlled ascents. The third 50 ft. casualty (No. 50) was a buoyant ascent. Doubt as to how well he exhaled was resolved by autopsy evidence of focal emphysema distal to a broncholith. Of all the accidents, it is definitely stated that 27 did not exhale, exhaled inadequately, or rose too quickly. This was especially characteristic of the S.E.A. casualties, all of whom used their appliances incorrectly, discarding the mouthpieces, and exhaling inadequately or not at all.

The onset of signs and symptoms occurred "within a few minutes" to 10 minutes after surfacing in 24 cases of aeroembolism. Of these, 4 relatively mild cases occurred within 3 to 10 minutes, 11 "upon Surfacing" or in less than one minute, and 9 at some level in the water (usually between 40 and 10 ft.). Of the 8 fatalities, 2 were struck while still ascending in the water, 4 at or just before the surface, and 1 within a few minutes after surfacing. As a group, they exhibited embolization earliest.

The symptoms and signs of the aeroembolism cases were predominantly those of central nervous system involvement. In order of frequency, these were: [A] unconsciousness or dizziness (32), focal paralyses (18), convulsions and incontinence (17), extra-alveolar air (pneumothorax, and mediastinal and subcutaneous emphysema) found on physical examination, X-ray, or autopsy, or very commonly indicated by chest (mostly substernal) pain (16), localized anesthesias, paresthesias, and pains (15), visual disturbances of complete or partial vision loss, eye muscle imbalances, and pupillary changes (7), focal pareses (4), signs of shock which might have a neurologic or cardiac origin (8), dyspnea of neurologic, cardiac, or pulmonic origin (6), 44

psychic changes (1), nausea and vomiting (1), and headache or "eye pain", mentioned a few times but probably present in more. The fatalities were all, of course, unconscious and eventually in shock. Five (5) had convulsions, 2 had subcutaneous emphysema, 1 had focal paralysis, and 1 had hemoptysis.

In those aeroembolism cases which survived, recompression was begun whthin an average of three minutes in 23 cases. In 7 cases, recompression was begun within six to ten minutes after the appearance of signs and symptoms. In one case (No. 29) treatment was begun after 1-1/2 hours, but this involved only progressive sensory changes in one hand, with substernal and precordial chest pain and may have been due to extra-pulmonary air dissecting in the chest and on to the brachial plexus. Another case (No. 11) treated after two hours, did survive, but had signs and symptoms for the following two days. Four (4) survivors (Nos. 5, 6, 7 and 10) received no recompression. One fatality (No. 4) was not recompressed, one was treated after 30 minutes (No.14), found unconscious, and perhaps dead at 35 ft.), 2 were treated within 5 to 6 minutes, and 2 within 2 minutes. These figures are significant in a consideration of the location of the treatment recompression chamber, and the time necessary to pressurize the victim.

The treatments have been somewhat variable. With the earliest casualties, little was understood of their etiology. Various theories and reports suggested accentuated effects of the Valsalva maneuver, central nervous system or fast tissue bends, or hysteria. From these evolved the animal experiments and resultant theories which related the casualties to air emboli resulting from excessive transthoracic pressure. Recompression would decrease the size of the obstructing bubble, allowing it to pass on or be absorbed. The earlier casualties were apparently recompressed to a depth of relief, with perhaps some added pressure as a margin of safety. Decompression from the treatment pressure was empirical or according to standard decompression tables; with the advent of the present standard U.S. Navy decompression and treatment tables in 1943, casualties began to be treated in a relatively standardized manner. Recompression was to a depth of 165 ft. (73.4 p.s.i.g.), sometimes deeper, with subsequent decompression over a 19 (table 3) to 37 (table 4) hour period, depending on how soon relief of signs and symptoms were obtained

Of the 36 air embolism survivors, 4 cases recovered without and recompression therapy, 1 with 15 lbs., 3 with 25 to 30 lbs, 6 with 40 to 45 lbs., and 2 with 60 to 70 lbs. of recompression for periods varying from 30 to 120 minutes. In an attempt to decrease the long treatment schedules of tables 3 and 4, table 2 was used successfully on 2 casualties (Nos. 22 and 23) in 1947, one of which recovered slowly (No. 22). Of the remainder, 15 survivors were treated initially on

table 3 and two on table 4 (to 180 ft. - Nos. 51 and 54). There were recurrences of aeroembolism manifestations in 4 cases. One (1) case (No. 11), treated with 25 lbs. for 10 minutes was not re-treated; 2 recurrences on table 3 (Nos. 37 and 47), both at 30 ft. during treatment, were re-treated successfully on table 4; the fourth case (No.56) recurred twice after initial treatment on table 3, each time being re-treated on table 4 (X-rays later revealed a lung lesion). Two (2) cases of aeroembolism which also had manifestations of extra-alveolar air had recurrences of the latter during treatment. They will be discussed later under extra-alveolar air casualties.

The first fatality (No. 4) was dead before recompression to 46 ft. (20 p.s.i.g.), and pressure was stopped after 10 minutes. The second fatality (No. 5) was not recompressed. Without pulse on removal from the water, he was given central nervous system stimulants in efforts to revive him. Although probably found dead in the water at 35 ft., the third fatality (No. 14) was recompressed to 80 p.s.i.g. for one hour. The record does not indicate presence of a pulse. Fatality four (No. 21), treated initially on table 4, continued to convulse despite sedatives, and died after an unrecorded time interval. The use of sedatives instead of further recompression to relieve the convulsions should be reconsidered in the evaluation of this apparent lack of response to pressure. The fifth fatality (No. 34) was taken to 165 ft., but was dead in less than one minute. It was recorded that O_2 was administered at 165 ft. for this case. Fatality six (No. 36) was recorded as dead before entering the chamber, but was nevertheless recompressed to 70 ft. before efforts at reviving him were discontinued. He had been recompressed within 2-1/2 minutes of arrival at the surface. Fatality seven (No. 45), entering the chamber 6 minutes after the onset of signs at 50 ft. on a 100 ft. free ascent, died after 4-1/2 hours on table 4. Fatality eight (No. 50), recompressed within 2 minutes after being struck at the surface, died between 165 and 220 ft. (2 minutes of recompression). Stimulants and artificial respiration were also used. Two fatalities (Nos. 21 and 45) lived, therefore, long enough to be treated on parts of table 4. The majority of deaths occurred within two minutes of the onset of manifestations of the aeroembolus.

Chest X-rays were taken after removal to a hospital, but demonstration of the etiological pathology was recorded on the NavMed 816's of only 3 cases (Nos. 56, 57 and 62). These three had all made normal ascents. Most of the cases with gross extra-alveolar air had X-ray evidence of this. The majority of the films taken may not have been read by physicians familiar with lesions or conditions of the lung caused by or liable to cause intra-alveolar over-pressurization during the rapid decompression of the ascents.

Autopsies were performed on all fatalities, with the exception of the most recent (No. 50); they were performed by physicians not completely familiar with the findings in a possible aeroembolus victim. The autopsy on fatality eight was performed by two pathologists, both meeting the above requirement (one a former diving medical officer). It was the only case recording microscopic findings. All autopsies revealed essentially most of the following findings: Intra and extravascular air in most of the tissues. Especial emphasis was placed on the lungs, heart, and blood vessels, and incorrectly in some, the brain. The lungs revealed gross hyperemia and hemorrhagic areas, with what the examiners termed "emphsematous changes". Subpleural blebs were found, and air was present in the loose connective tissues surrounding the bronchovascular trees. In some cases it was found dissecting up into the mediastinum, and on into the subcutaneous tissues of the chest and neck. Minimal pneumothorax was found in at least one case (No. 50), which also had gross evidence of entrapped air. Air was present in the left chambers of all hearts, and in the right side of one (No. 50) with patent foramen ovale. All had coronary artery air bubbles. All of the brains examined had air in the superficial and deep arteries and veins. The eighth case (No. 50), on microscopic section, was noted to have dissection of a portion of the cerebrum by extra-vascular bubbles. Due to the methods of dissection, injection, and removal of brains, as well as post-mortem changes, intravascular bubbles in cadavers are not necessarily antemortem ones. Because of the uncertain time intervals before death, as well as the obviously complicating factors of recompression during treatment and subsequent post-mortem decompression, no definite conclusions can be drawn as to the origin of most of the intra-and extra-vascular bubbles. Some methods of collection and analysis of the gas might provide information on future fatalities, but post-mortem changes with decomposition would introduce further complicating factors.

Extra-alveolar air: Of the 60 escape training casualties, 22 had the signs and/or symptoms of extra-alveolar air present as mediastinal and subcutaneous (including cervical) emphysema. Two of these also had pneumothorax. Sixteen of these cases occurred in air embolism casualties, six occurred as separate entities. There were no deaths known to be due directly to the extra-alveolar gas, although two aeroembolism fatalities (No. 14 and No. 36) had this entity grossly visible before death.

With the exception of 3 who had undergone extensive past training, these casualties had made no ascents prior to the day of their accident. The number of ascents made on the day of, or previous to the appearance of ectopic air in the tissues averaged about three, and ranged from 0 to 6. The depths of these varied from 18 ft. to 110 ft. The depths of the last ascent before appearance of the signs and symptoms were: one

from 18 ft., 2 from 35 ft., 14 from 50 ft., and 5 from 100 ft. The methods of escape of those 16 occurring with aeroembolism have been noted above. Of the remaining 6, there was one 50 ft. S.E.A. ascent with apparatus used correctly, one 100 ft. S.E.A. ascent with breath held the last 10 ft., and four 50 ft. free ascents; of the latter four, two were normal ascents, one had difficulty with a prior 100 ft. free ascent and ran out of air at 50 ft. for 10 ft. (held breath), and the other (No. 62) had X-ray evidence of pulmonary histoplasmosis with a positive histoplasmin skin test (he had made a normal ascent).

The times of onset of signs and symptoms in those occurring with aeroembolism were immediately or less than 3 minutes after surfacing in 11 cases, 10 minutes after surfacing in one case, and during ascent in 4 cases. In the six cases occurring alone, 3 had their onset on or before surfacing, or a few minutes thereafter, (Nos. 24 and 50), two had a very mild onset shortly after surfacing, with progression to an obviously pathological condition later in the day, and one (No. 27) had the onset on the following day. The signs and symptoms of extra-alveolar air were, in order of frequency: substernal or chest pain (19 cases-2confirmed by X-ray, although other probable confirmations were not noted on accident reports), subcutaneous (including cervical) and mediastinal emphysema (10), sore throat, voice changes and dysphagia, occurring with other more obvious gross evidences of tissue air (3), hemoptysis in concurrent aeroembolism (2), and pneumothorax (2). Substernal or chest pain was considered evidence enough of some intra-mediastinal air even though clinical examination or X-ray did no reveal it grossly. In a review of pre and post-ascent chest X-ray on 100 buoyant ascents, British physicians noted subclinical mediastinal emphysema in one of the first 70 cases. The actual incidence of such an occurrence is only speculation.

The time interval between appearance of clinical signs and symptoms of extra-alveolar air in those cases without aeroembolus, and beginning of recompression therapy was: immediately in one case, 1-1/2 hours in another, 3 hours in a third, and 12 hours in a fourth case. Two (2) cases (No. 27 and 62) were not recompressed.

The treatment of the extra-alveolar air present in the 16 cases of aeroembolism was, by necessity, the treatment of the more serious intravascular air. This is listed above under aeroembolism treatment routines. Two (2) of these cases, both survivors, had recurrences of extra-alveolar air during treatment. One (1) case (No. 19) had relief of his embolus at 50 ft., but with recurrence of chest pain, and he was recompressed on table 1 (this was primarily an aeroembolus case, occurring one year after introduction of standard treatment tables). He again had recurrence of chest pain, with pneumothorax appearing during decompression. Increasing mediastinal emphysema led to a superior vena syndrome, and this second

recurrence was unsuccessfully treated with O at 20 ft., the pneumothorax was reduced via syringe. Another case of aeroembolism (No.29) had recurrence of previous chest pain on ascent during treatment table 3, but was not re-treated.

Of the 6 cases of extra-alveolar air without aeroembolism, 2 were not recompressed. One (1) of these (No. 27) had subcutaneous and mediastinal emphysema with onset on the day after the ascent; the other (No. 62), an individual with X-ray evidence of old histoplasmosis lesions (calcified), who had onset of substernal pain immediately after ascent. Three (3) cases were treated, without recurrence, on table 3. A sixth case (No. 24) was treated with 50 p.s.i.g. (table 1 ?), but had a recurrence the next day which was re-treated successfully on table 3. His original manifestations of subcutaneous and mediastinal emphysema had occurred later in the day of the ascent.

As noted above, X-ray evidence was recorded in only 3 cases of extra-alveolar air, although other probable confirmations were not on the reports. To these must be added the one subclinical case, previously mentioned, at Gosport. Even pre- and post-ascent chest X-rays on all escapees will not reveal every case of extra-alveolar air (as in the case of No. 62).

3. Miscellaneous: Of the 60 casualties resulting from escape training water work, 11 were not due to over-pressurization of intra-alveolar gas. Eight (8) of these were cases of decompression illness (6 in instructors), one was due to ear injury (No. 42), one pulmonary "squeeze" (No. 43), and one hysterical syncope (No. 59). Because of their particular etiology, these cases will not be examined in great detail, nor will they be considered statistically to be a direct result of submarine escape ascents, per se.

All cases of decompression illness had obviously overexposed themselves to air under pressure through repeated dives, or, in some cases, recompression chamber tending after a day of escape tank work. The necessity for adequate decompression after multiple dives was not considered in most, and where it was, the decompression was insufficient.

In 3 cases, the onset of bends was 1-5 minutes after surfacing from the last dive, in 5 cases, the onset was 2-5 hours later.

With the exception of one case of skin bends (immediate onset) all had the characteristic pain in upper and/or lower limbs.

The recompression therapy, in every case table 1 was started after 1/2 to 5 hours' time from onset of symptoms and signs. There were no recurrences or fatalities.

One case (No. 42), had dizziness, "crackling" sensations in his left ear and right lateral nystagmus upon surfacing from a 45 ft. free ascent. He had made three good 50 ft. free ascents previously that day. Within 3 minutes he was recompressed to follow table 3, but, since there was little alleviation of complaints, he was switched to table 2 and described as a case of possible vestibular injury.

One minute after surfacing from a 100 ft. skin dive, a former tank Officer in Charge, (No. 43) experienced substernal pain, dyspnea, and hemoptysis. This was his first "bottom drop" skin dive in the tank, and his probable thoracic "squeeze" (lung compression beyond residual volume) was not actively treated. Chest X-rays were negative.

Another trainee (No. 59), after a normal 50 ft. buoyant ascent (a previous 18 ft. buoyant ascent was also normal), fainted at the surface. He was recompressed within 2 minutes to 165 ft., and at no time exhibited signs of neurological damage. He had regained consciousness before 165 ft. in the chamber, and complained only of dizziness and slightly blurred vision when erect, subsiding when recumbent. This was diagnosed as primary syncope, probably due to anxiety reaction or hysteria, even though the man appeared stable emotionally. He was decompressed on standard Navy decompression tables with no further difficulties. The number of cases of primary syncope, without other neurological signs, which may have been misdiagnosed as aeroembolism and treated as such (supposedly "successfully") can only be a matter of conjecture. The difficulty, at times, of the clinical diagnosis of aeroembolism in such cases can only lead to a rule that all of them must be treated on standard treatment tables 3 or 4. This to avoid the possibility of a fatality or permanent damage.

D. Clinical Characteristics of Recompression Chamber Casualties

1. Introduction and etiological factors:

In addition to the casualties occurring in the water phase of training, there are recorded 17 cases of recompression chamber casualties.

Every trainee receiving submarine escape training is required to first undergo a pressure test of 50 p.s.i.g. (112 ft.). Aside from obvious differences in milieu, the pressure test has important similarities to the water training. A trainee holding his breath or one with a lung containing an entrapped air space, will develop the same increase in intra-alveolar pressure as a trainee on ascent in the water. The difference is that in the dry chamber, the individual is respiring freely, whereas in the water he obviously can only be exhaling.

2. Air Embolism

Of the 17 chamber casualties, there were 10 cases of aeroembolism, none of which was fatal. Only one of these was specifically noted to have held his breath on ascent.

For all these cases it was their first exposure to pressure, with the exception of one who failed to equalize ear pressure on a similar test one week previously.

Where recorded, the onset of the signs and symptoms of air embolism was at 10 to 20 ft. on the chamber ascent in 3 of the cases, upon surfacing in 2, and within a few minutes of surfacing in 3 cases.

The signs and symptoms were similar to those of the previously described water training aeroembolism cases. In order of frequency, they were: paralysis and/or paresis (5), convulsions (4), dizziness and/or vertigo (4), sensory changes such as anesthesias, or pain (4), visual disturbances of loss of vision, eye muscle imbalances, etc. (4), unconsciousness (3), extra-alveolar air (2), nausea and vomiting (2), deafness and tinnitus (1), skin rash (1), headache-mentioned specifically in only one case, but probably present in many more. Here, more-so than with the aeroembolism casualties of water ascents, there was difficulty in making a differential diagnosis with possible decompression illness. The signs and symptoms in 2 cases (Nos. 65 and 77), consisting mostly of sensory changes with some weakness, might have been diagnosed as decompression sickness, except that the onsets occurred so quickly (before or at the surface) after a "dive", with bottom time for that depth being less than the no-decompression limit.

Where recorded, the time interval between onset of aeroembolism and treatment was: in 2 cases 1-2 minutes, in two 10-13 minutes, and in two about 30 minutes. The delays were probably due more to the lack of or difficulty in making a diagnosis, since all of these cases had an almost immediate onset (see above).

The initial treatment of the intravascular air was consistent: 9 cases treated on table 3, one on table 4. One (No. 64) was recompressed to 165 ft. (73 p.s.i.g.), and no table recorded, but table 3 is assumed. There were two recurrences: one (No. 76) at 40 ft. on table 3, successfully retreated on table 4; the other (No. 77), a recurrence of convulsions and unconsciousness after table 3, required both tables 3 and 4 for successful further treatment.

No X-rays were recorded on any of these casualties, although they were probably taken on most and recorded in their health records.

3. Extra-alveolar air: There were 4 cases of extra-alveolar air extravasation, two (Nos. 70 and 79) of which occurred in cases of air

embolism. Of these latter two, one had the symptoms of interstitial air 5 minutes after aeroembolism occurred, the other almost immediately. Of the two isolated extra-alveolar air cases, (Nos. 63 and 75), one had an onset at 10 ft. on the chamber ascent, the other (as with some of the water casualties) later in the day.

The symptoms and signs, in order of their frequency, were: subcutaneous (including cervical) emphysema (3), mediastinal emphysema, including substernal or chest pain or fullness (3), voice changes accompanying gross interstitial air (3), cyanosis (1 case), and dysphagia (1 case).

In the two cases occurring with aeroembolism, treatment occurred in about 10 minutes on table 3, without recurrence. The one case (No. 75) with delayed onset later in the day, after the chamber run, was treated on table 3 without recurrence. One case (No. 63) received no treatment.

No X-rays were recorded on the NavMed 816's of these casualties.

4. Miscellaneous: Although there were two cases of aeroembolism whose signs and symptoms suggested decompression sickness, there was only one clear-cut case of the latter, (No. 69). This occurred in an instructor who made a chamber test (table 3) on a trainee (with chest pains later found not to be due to a diving accident) in the evening after a day of tank work. Symptoms of skin and limb bends with minor visual disturbances began one hour after surfacing and were successfully treated on table 2. Four cases of miscellaneous, minor disorders occurred during chamber work. Two of these were hysteria, although many more unrecorded cases of this have probably occurred. One (No. 66) happened during compression (before ascent), and consisted of subjective numbness, paralysis, and dizziness; the other (No. 74), had dizziness, numbness and nausea after epistaxis at 25 ft. on ascent. The former was treated on table 3, the latter on an "abbreviated" table 3. One trainee (No. 67) noted his right arm "warmer" than the left after a chamber run, and was treated on table 1, with questionable relief. Another case (No. 68) with questionable visual disturbances and an allergic-type rash, received no treatment, and made subsequent pressure tests and ascents without incident.

E. Statistical Analysis of Escape Training Tank Casualties

1. Introduction

A complete statistical analysis of all casualties and the various methods of individual submarine escape training is impossible due to the unavailability of most of the necessary records from the Pearl Harbor and British escape training tanks.

It is conservatively estimated that approximately 250,000 escapes of all types have been made in the three tanks since the inception of training. This does not, of course, include the routine daily escapes made by instructors working in the water, which would increase that figure many times. Also not included are recompression chamber pressure "runs", a record of the number of individual chamber runs never having been kept.

2. Incidence of Casualties: The incidence of air embolism and extra-alveolar air casualties and fatalities during water training is as follows:

a. Aeroembolism:

Casualties/total ascents = $\frac{44}{250,000}$ = .018%

Fatalities/total ascents = $\frac{8}{250,000}$ = .003%

Fatalities/total casualties = $\frac{8}{44}$ = 18.2%

b. Extra-alveolar air:

Casualties/total ascents = $\frac{22}{250,000}$ = .009%

(No fatalities)

3. Incidence of water casualties by year and method of ascent:

1928	S.E.A.	-	1	
1930	S.E.A.	-	3	(1 fatality)
1931	S.E.A.	-	7	(1 fatality)
1933	S.E.A.	-	1	
1934	S.E.A.	-	2	(1 fatality)
1938	S.E.A.	-	1	
1942	S.E.A.	-	2	
	F.A.	-	1	
1944	S.E.A.	-	1	
1947	S.E.A.	-	1	(1 fatality)
	F.A.	-	2	
1948	F.A.	-	1	
1950	F.A.	-	2	
1951	F.A.	-	3	
1952	F.A.	-	4	(2 fatalities)

1953	S.E.A.	-	1	
	F.A.	-	1	
1954	F.A.	-	2	(1 fatality)
1955	F.A.	-	3	
1956	F.A.	-	1	
1957	F.A.	-	3	(1 fatality)

There is no significant correlation between the number of ascents per year and the incidence of casualties and fatalities. This is shown by comparison of the above with figures of total ascents in the New London tank (representative of the workload at all 3 tanks) - (See Page 96, MRL Report No. 264). (See Page 23 appended).

4. Chamber casualties are not included in the above figures, since for statistical purposes the total number of chamber runs is not known. Only air embolism and extra-alveolar air casualties are presented, since only they are considered to be a direct result of the actual method of ascent.

5. Escape training experience at the New London tank:

a. Only the New London Submarine Escape Training Tank has maintained careful records of the total number of ascents, including the various methods, with files of the casualties. It is the oldest and busiest tank, putting through new recruits as well as re-qualifiers, all of which makes its records statistically more significant.

b. Statistics of the New London tank, using the three methods of individual submarine escape are as follows:

Air embolism and extra-alveolar air: 30 cases, 4 of which were fatalities (aeroembolism)

	Casualties Per Total No. Ascents	Fatalities Per Total No. Ascents	Fatalities Per Total Casualties
S.E.A.	12/193,000 = .006%	1/193,000 = .0005%	1/12 = 8%
Free Ascent	16/16,500 = .10%	2/16,500 = .012%	2/16 = 12.5%
Buoyant Ascent	2/6,500 = .03%	1/6,500 = .015%	1/2 = 50%
All types of Ascents	30/216,000 = .014%	4/216,500 = .002%	4/30 = 13.4%

6. Open sea training:

Because of incomplete records, no statistical analysis of open-sea escapes can be given, either as training or under actual submarine bottoming conditions.

F. Summary:

An analysis of all known casualties and fatalities occurring as a result of submarine escape training has been presented; this has included a detailed clinical discussion of the individual escapes, including the methods used, the manifestations of injury, and the treatments given. Statistics have been compiled to illustrate the incidence of the casualties and fatalities, and their relationships to the various methods of individual submarine escape.

TABLE 1 - Number and Kinds of Escapes and Associated Casualties

Fiscal Year	ESCAPES Total	S.E.A.	Free	Buoyant
1930	1299	1299		
1931	1138	1138		
1932	950	950		
1933	955	955		
1934	472	472		
1935	622	622		
1936	719	719		
1937	1555	1555		
1938	1445	1445		
1939	1713	1713		
1940	3831	3831		
1941	8257	8257		
1942	16359	16359		
1943	26970	26970		
1944	3021	3021		
1945	3231	3231		
1946	2094	1649	445	
1947	7022	6618	404	
1948	8698	8431	267	
1949	7480	6987	493	
1950	19718	16335	3383	
1951	25559	19158	6401	
1952	16686	15739	947	
1953	6353	5563	785	
1954	14726	12866	1860	
1955	19512	18538	974	
1956-57	18943	11958	438	6555
1930-1957	216,500	193,004	16,397	6555

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APPENDIX A

SUBMARINE ESCAPE TRAINING CASUALTIES - WATER TRAINING

Casualty Codes: A-Aeroembolism S-Extra-Alveolar Air B-Decompression Illness P-Pneumothorax M-Miscellaneous X-Fatality

NO.	CASUALTY	NAME	DATE	LOCATION	PAST	ASCENTS IMMEDIATELY PRECEDING	ACCIDENT	WHERE & WHEN DISCOVERED	SIGNS AND SYMPTOMS	HOW SOON TREATED	RX TABLES	SEQUELAE, ETC.	REMARKS	MD*
1	A	L., J.	8/15/28	Mine Tank, EDU-NGF			60 ft SEA	Few minutes after surfacing	Unconscious, dyspnea, paralysis	3 minutes	30# for 48 min.	Recurrence of paresis and headache after 45 minutes. Relief after second recompression to 25#	Fast ascent from 10 ft.	1
2	A	R., J.R.	after 8/30/30	N.L.		18 ft. SEAx2 (good)	50 ft. SEA	Few minutes after surfacing	Convulsions, Unconscious, shock		30 ft. (15#)		Fast ascent from 30 ft.? Held breath and did not use SEA	2
3	A	M., H.	9/12/30	Mine Tank, EDU-NGF			30 ft. SEA	Few minutes after surfacing	Unconscious, shock, paralysis, anesthesia	6 minutes	40# for 35 min.	Headache	Held breath and did not use SEA	1
4	AX	A., W.	before 10/30/30	P.H. (bell)		5 ft. SEA 10 ft. SEA 20 ft. SEA	30 ft. SEA	Few minutes after surfacing	Unconscious		46 ft. (20#) for 10 minutes	Dead on recompression	Autopsy: aeroembolism	3
5	AX	M., C.A.	5/?/31	U.S.S. Ortolan San Diego		7-1/2 ft. SEA (good)	15 ft.	Upon surfacing	Unconscious, shock		No recompression	Dead on removal from water	Autopsy: aeroembolism	4
6 7 8	A(x3)	Three cases at P.H.-before July, 1931. All had manifestations after 30 ft. SEA. Onset of several minutes after surfacing: cramps (abdom. and musc.), unconsciousness, and dyspnea. All survived, but one had immediate relief after recompression to 40#												3
9	A	K., L.S.	before 7/?/31	Mine Tank EDU-NGF			35 ft. F.A.	Upon surfacing	Dyspnea, unconsciousness, paresthesia, shock		45# for 120 min.	Recurrence of headache & weakness in 1 hr & recompressed to relief (?)	Defective SEA, did F.A. and failed to exhale	1
10	A	B., W.J.	before 7/?/31	N.L.			18 ft. SEA	Few min. after surfacing	Unconsciousness, convulsions, shock, dyspnea		No recompression. Revived in 15 min.		Fast ascent fm 10 ft. Held breath and did not use SEA	2
11	A	L., V.	before 7/?/31	N.L.	At least 2 yrs. experience as instructor	18 ft.SEA(fast) 18 ft.SEA(good) 50 ft.SEA fast from 10 ft.	50 ft. SEA	1/2 to 1 min. after surfacing	Shock, convulsions, visual signs, anesthesia	2 hrs.	25# for 10 min. then stopped. Symptoms & signs present for next one to two days		Fast ascent from 40 ft. Held breath	2

*SEE M.D.'S CODE ON LAST PAGE.

NO.	CASU-ALTY	NAME	DATE	LOCA-TION	PAST	ASCENTS IMMEDIATELY PRECEDING	ACCI-DENT	WHERE & WHEN DISCOVERED	SIGNS AND SYMPTOMS	HOW SOON TREATED	RX TABLES	SEQUELAE, ETC.	REMARKS	MD*
12	A	R., R.J.	10/2/33	P.H.	18 ft. SEA 100 ft. SEA in past 2yr		100 ft. SEA	Few min. after surfacing	Unconsciousness, convulsions, mouth bleeding	2 min.	40# for 45 min.	Epigastric distress convulsions, 30 min. after initial Rx. Recompressed to 175 ft. (77#) for 10 min. and decompressed for 76 min. (double decomp. on all stops).	Did not use SEA above 30 ft. ? Breathed out all the way	5
13	A	W., P.L.	1/12/34	N.L.	50#chbr 12 ft. SEA (x2) 18 ft. SEA (x2) 50 ft. SEA (x2)		100 ft. SEA	Within one minute after surfacing	Unconsciousness, shock	few min (?)	70# for 10 min. 4-1/2 hr. decompression	Leg bends on recompr.	Breathing in lung difficult at 50 ft and 30 ft then fast ascent to surface	3
14	ASX	G., I.R	7/27/34	N.L.			100 ft. SEA	In water at 30-40 feet	Unconsciousness, subcutaneous emphysema	30 minutes	80# for one hour	Heart stopped after one hour	Unconscious in water at 30-40 ft ? dead in water ? held breath Autopsy: aeroembolism	3
15	A	L., L.F.	8/2/38	N.L.	"several" in previous years	50# chbr 12 ft. SEA	18 ft. SEA	1-2 minutes after surfacing	Paralysis, visual disturb., dilated pupils	few minutes ?	60#	Improvement good, but recurrence of headache on decompression and recompression, to 45# with relief	Held breath all the way up. Did not use SEA (resistance in valves) lung found o.k.	6
16	A	P., A.W	7/11/42	N.L.	Instructor	2-100 ft. dives in 2 hrs. for TOB of 32 min.	100 ft. F.A.	3 minutes after surfacing	Pain, numbness, and paresis of ft. lower limb	3 min.	45# for 30 min.		Instructor, and this was possibly decompression illiness (110 ft. for 32 min.)	7
17	A	G., G.L.	8/7/42	N.L.			50 ft. SEA	At 18 ft. on ascent	Shock, unconsciousness	(?)	45# (?)		Lost mouthpiece at 35 ft. and made F.A. from 35 ft. to 18 ft. Held breath and did not use SEA	7
18	AS	H., E.B.	9/12/42	N.L.	Previous ascents successful (3)		50 ft. SEA	1 minute after surfacing	Unconsciousness, convulsion, substernal pain eye disturbances	(?)	45# and then to 60#		Difficulty on last 18 ft. Made fast ascent without using SEA. Held breath.	7

*SEE M.D.'S CODE ON LAST PAGE.

).	CASU-ALTY	NAME	DATE	LOCA-TION	PAST	ASCENTS IMMEDIATELY PRECEDING	ACCI-DENT	WHERE & WHEN DISCOVERED	SIGNS AND SYMPTOMS	HOW SOON TREATED	RX TABLES	SEQUELAE, ETC.	REMARKS	MD
9	ASP	C., J.S.	1/27/44	N.L.	50# chbr	12 ft.SEA (x2) 18 ft.SEA (x2)	50 ft. F.A.	On surfacing	Unconsciousness, hemoptysis, incontinent, substernal soreness, pneumothorax	less than 3 min.	50#	Relief, but recurrence of chest pain and recompressed on table 1. Again had chest pain and partial left pneumothorax on decompression. Mediastinal emphysema with SVC syndrome, treated unsuccessfully with O_2 at 20 ft. Reduced by syringe suction.	Difficulty in breathing from 20 ft. to surface & held breath. Did not use SEA	8
0	B	W., W.J.	1/26/46	NMRI	150 ft. pressure previous day		100 ft. SEA	5 minutes after surfacing	Lt. axillary & scapular pain	22 min.	Table 1		TOB 4 min, using O_2 and air	9
1	AX	S., E.C., Jr.	3/22/47	P.H.			100 ft. SEA (O_2)	unconscious at surface	Unconsciousness, convulsions	(?)	Table 4	Sedative without effect	Lost mouthpiece, held breath, & ascended without SEA	10
2	AS	H., R.R.	6/16/47	N.L.		12 ft. F.A.(x2) 18 ft. F.A.(x2) 35 ft. F.A.	35 ft. F.A.	Few min. after surfacing	Flaccid, unconsciousness, hemoptysis, shock, incontinent subcutan. cervical and mediastinal emphysema (substernal soreness)	3 min.	Talbe 2	Slow recovery	Did not exhale above 10 ft., and swam up	11
3	AS	Y., J.C.	12/18/47	N.L.	Prospective instructor, qualified to 18 ft. F.A.	25 ft. F.A.(x2) 18 ft. F.A.	*90 ft. to 50 ft. 50 ft. to 25 ft. 25 ft. to surface F.A.	50 ft. lock to surface	Numbness at surface, paralysis convulsions at 50 ft.substernal pain, subcutan. cervical and mediastinal emphysema	7 min.	Table 2	(experiment)		7 12
4	S	C., P.F.	9/23/48	N.L.	(?)	50# chbr. 10 ft SEA (X2) 18 ft. SEA (2).50 ft. SEA	50 ft. SEA	Later in the day, but complained earlier	Meningismus, disorientation, eye musc. disturbance, epigastric pain, subcutaneous emphysema, substernal pain, sore throat	3 hrs.	50#	Recurrence next day, with successful recompression on table 3	Symptoms noted later in day. ?Mediastinal emphysema	7
25	AS	C., T.M.	9/27/48	N.L.			50 ft. SEA	Few minutes after surfacing	Right hand numbness and paralysis, chest soreness	2 min.	Table 3		Normal ascent	18

SEE M.D.'S CODE ON LAST PAGE.

NO.	CASU-ALTY	NAME	DATE	LOCA-TION	PAST	ASCENTS IMMEDIATELY PRECEDING	ACCI-DENT	WHERE & WHEN DISCOVERED	SIGNS AND SYMPTOMS	HOW SOON TREATED	RX TABLES	SEQUELAE, ETC.	REMARKS	MD
26	A	C., R.I.	8/28/50	N.L.		8 ft. F.A.(X2) 15 ft. F.A.(X2) 25 ft. F.A.(X2) (good)	40 ft. F.A.	30 seconds	Flaccid and rigid paralysis	3-4 min.	Table 3		Insufficient exhalation	19
27	S	S., P.W.U.	9/6/50	N.L.		18 ft. SEA 50 ft. SEA 100 ft. SEA(X2)	100 ft. SEA	Next day	Precordial pain with breathing, subcutaneous emphysema		No recom-pression		Held breath at 10 ft. on last 100 ft. ascent, and did not use SEA	2
28	AS	K., A.J.	10/11/50	N.L.		18 ft. F.A.(X3) (good)	50 ft. F.A.	18 ft. lock, after 1 min.	Paralysis, Un-consciousness, anesthesia, chest pain, subcutaneous and mediastinal emphysema	4 min.	Table 3		Ascended faster than bubbles	20
29	AS	P., L.M.	1/30/51	P.H.		50# chbr 18 ft. F.A. (good)	18 ft. F.A.	10 min. after surfacing	Progressive anesthesia of hand, substernal pain	1-1/2 hrs.	Table 3	Chest pain after recompression	Fast ascent from 18 ft. (?)	21
30	A	G., J.F.	3/30/51	Petrel			50 ft. F.A.	At surface	Unconsciousness	?	Table 3			42
31	B	C., W.A.	5/18/51	N.L.	Instructor		Skin diving to 40-50 ft for 90 min. and then F.A.	2-1/2 hrs. after sur-facing	Pain in left foot, and skin bends	1/2 hr	Table 1		50 ft., with TOB 90 minutes	19
32	B	L., J.	5/21/51	N.L.		Repeated skin Dives 0-100 ft. plus 100 ft. F.A.		5 minutes after sur-facing	Pain in left knee	5 hrs. after onset	Table 1			19
33	A	T., D.E.	7/12/51	Petrel			12 ft. F.A.		Paresis, dizzy, paralysis, anesthesia, convulsions	?	Table 3			42
34	AX	B., M.G.	2/5/52	N.L.	100 ft. SEA three yrs. before 18 ft. SEA 50 ft. SEA 100 ft. SEA (day before all good)	18 ft. F.A.(x3)	50 ft. F.A.	Upon surfac-ing	Unconsciousness, convulsions, Shock, nemoptysis	5 minutes	To 165 ft. but dead in 1 min. Given 02 at 165 ft.		Autopsy: aeroembolism	43
35	S	M., R.B.	6/6/52	N.L.		18 ft. F.A.(x3)	50 ft. F.A.	at 18 ft.	Chest pain at 18 ft. with subcutaneous emphysema	?	Table 3			44

*SEE M.D.'S CODE ON LAST PAGE.

NO.	CASU-ALTY	NAME	DATE	LOCA-TION	PAST	ASCENTS IMMEDIATELY PRECEDING	ACCI-DENT	WHERE & WHEN DISCOVERED	SIGNS AND SYMPTOMS	HOW SOON TREATED	RX TABLES	SEQUELAE, ETC.	REMARKS	MD*
36	ASX	C., L.	7/1/52	N.L.		18 ft. SEA, 50 ft. SEA, 100 ft. SEA, 18 ft. F.A. (x3)	50 ft. F.A.	Upon surfacing	Convulsions, unconsciousness subcutaneous emphysema, dead before entering chamber	2-1/2 minutes	To 70 ft. Dead before recompressed		Autopsy: aeroembolism	13
37	AS	M., R.P.	8/5/52	N.L.		50 ft. F.A. (good)	100 ft. F.A.	2-3 minutes after surfacing	Psychic changes substernal pain paresis, spastic	5-10 minutes	Table 3	Recurrence at 30 ft. and recompression or Table 4	Ascended too fast, insufficient exhalation	13
38	S	A., J.D.	12/11/52	N.L.		100 ft. F.A.	50 ft. SEA	Few minutes after surfacing	Fullness in neck, voice change, tension and fullness in chest, subcutaneous and mediastinal 3mphyw3ma (x-ray)	1-1/2 hours	Table 3		Had difficulty with previous F.A. from 100 ft. when ran out of air at 50 ft. and held breath for 10 ft. (?)	13
39	A	G., D.D.	12/11/52	N.L.		100 ft. F.A. (good)	50 ft. F.A.	At 40 ft. on ascent, and 5 minutes after surfacing	Dizziness, pupil inequality	6 minutes	Table 3			13
40	AS	E., D.R.	4/13/53	P.H.		18 ft. F.A. 35 ft. F.A. 50 ft. F.A.	50 ft. F.A.		Arm and leg, substernal pain, visual disturbances, dizziness. Rales in right lung base posteriorly	?	Table 3			14
41	AS	D., N.G.	5/11/53	N.L.	Two other qualifications in past	18 ft. SEA 50 ft. SEA	100 ft. SEA(O_2)	At 20 ft. on ascent	Legs and hands paralyzed, anesthesia, mediastinal emphysema	6 min.	Table 3		Held breath, did not use SEA, and did F.A.	15
42	M	J., J.C.	11/9/53	N.L.		50 ft. F.A. (x3) (good)	45 ft. F.A.	15 seconds after surfacing	Dizziness, "crackling" in right ear, nystagmus to right	3 min.	Table 3 switched to table 2		Probably vestibular disturbance	16
43	M	P., J.H.	3/2/54	N.L.			100 ft. skin dive	One min. after surfacing	Substernal pain, dyspnea, hemoptysis		No Rx		First "bottom drop". Thoracic squeeze	17
44	B	H., R.F.	3/8/54	N.L.	Instructor, many dives to 70 ft.			2-3 hrs. after work	Right lower extremity pains	3 hrs	Table 1		Previously had pains in that extremity	13

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NO.	CASU-ALTY	NAME	DATE	LOCA-TION	PAST	ASCENTS IMMEDIATELY PRECEDING	ACCI-DENT	WHERE & WHEN DISCOVERED	SIGNS AND SYMPTOMS	HOW SOON TREATED	RX TABLES	SEQUELAE, ETC.	REMARKS	MD*
45	AX	D., W.S.	4/20/54	P.H.		12 ft. F.A. 18 ft. F.A. 50 ft. F.A.	100 ft. F.A.	At 50 ft.	Convulsions, unconsciousness paralysis	6 min.	Table 4	Death after 4-1/2 hours	Autopsy: aeroembolism	14
46	B	F., J.L.P.	9/14/54	P.H.	Skin diving at 100 ft, 70 ft, 50 ft. 25 ft. and 10 ft. levels			On surfacing	Mild skin bends	?	Table 1			14
47	A	A., C.F.	10/15/45	P.H.		50 ft. F.A. (good)	100 ft. F.A.	On surface	Unconsciousness, convulsions, nauseated and vomiting	30 sec.	Table 3	Recurrence at 30 ft. and recompression on table 4		22
48	B	B., G.F.	10/17/54	P.H.	Instructor				Bilateral knee pains after treating embolism case on table 3, with recurrence on table 4	3 hours	Table 2			23
49	AS	E., R.H.	4/19/55	P.H.		?	50 ft. F.A.	10 ft.	Dyspnea, chest pain, dizziness	?	Table 3		Ran out of air at 10 ft, held breath	22
50	S	G., R.E.	4/27/55	N.L.		18 ft. F.A.(x2) (good)	50 ft. F.A.	Later in day	Neck and chest pain, dyspnea, hoarseness, dysphagia, mediastinal and subcutaneous emphysema	12 hours	Table 3		Symptoms of extra-alveolar air appeared later in day	24
51	A	H., R.A.	6/8/55	N.L.		18 ft. F.A.(x2) 50 ft. F.A. (good)	50 ft. F.A.	Few minutes after surfacing	Convulsions, unconsciousness paralysis, anesthesia	2 minutes	Table 4 (to 180 ft.)	Recurrence after 5 hrs with weakness. Given HeO_2	Fast ascent above 30 ft.	24 et al
52	AS	W., R.A., Jr.	9/9/55	U/W SS Key West	18 ft. F.A. (good)		35 ft. F.A.	One minute after surfacing	Dizziness, anesthesia, chest pain, paralysis, visual disturbance	1 to 5 minutes	Table 3		Fast ascent on last 10 ft.	25 or 26 (?)
53	B	D., J.M.	11/10/55	N.L.		18 ft. SEA 50 ft. SEA 100 ft. SEA		5 hours	Bends (?)	7-1/2 hours	Table 1A			27

* SEE M.D.'S CODE ON LAST PAGE.

NO.	CASU-ALTY	NAME	DATE	LOCA-TION	PAST	ASCENTS IMMEDIATELY PRECEDING	ACCI-DENT	WHERE & WHEN DISCOVERED	SIGNS AND SYMPTOMS	HOW SOON TREATED	RX TABLES	SEQUELAE, ETC.	REMARKS	MD *
54	A	S., F	12/11/55	N.L.	Instructor		85 ft. F.A.	2 minutes	Unconsciousness, chest pain	10 min.	Table 4 (to 180 ft.)		Free ascent from 85 ft. to 50 ft. with insufficient exhalation. Dropped to 100 ft and then rose to 85 ft. again where became unconscious.	24
55	AS	S., E.C.	12/4/56	N.L.		50 ft. F.A. (good)	110 ft. F.A.	Few minutes after surfacing	Chest pain, paralysis, anesthesia	1 min.	Table 3		Did not exhale from 110 ft. to 100 ft. Slow exhalation all the way up.	28
56	A	A., C.H.	2/7/57	P.H.		18 ft. B.A. 50 ft. B.A. (good)	50 ft. B.A.	3 minutes	Headache, paresis paresthesia, visual disturbance, convulsions, unconsciousness	7 min.	Table 3	Two recurrences, both treated on table 4, hemoptysis later	X-ray evidence of pre-existing right lower lobe lesion (cyst?)	29
57	AS	S., D.L.	3/8/57	N.L.		18 ft. B.A. (good)	50 ft. B.A.	During ascent and few minutes after surfacing	Chest pain on ascent. Unconsciousness, convulsions, hemophysis,? mediastinal emphysema	2 min.	Table 3		LLL bleb over diaphragm, on x-ray	26
58	B	S., J.E.	3/8/57	N.L.		Limb pains after treating embolism case on table 3. Water work previously.				3 hours	Table 1			26
59	M	P., J.L.	4/1/57	N.L.	50# chbr (good)	18 ft. B.A. (good)	50 ft. B.A. (good)	At surface	Syncope, visual disturbances	2 min.	Standard decompression from 165 ft. (170 ft. for 40 min.)		Hysteria with primary syncope	30
60	AX	H., R.L.	4/2/57	N.L.	?	18 ft. B.A. 50 ft. B.A. (good)	50 ft. B.A.	At surface	Unconsciousness, convulsions	1-2 min.	To 220 ft death between 165 and 220 ft. (2 minutes)		Autopsy: aeroembolism due to entrapped air space	23

* SEE M.D.'S CODE ON LAST PAGE.

NO.	CASU-ALTY	NAME	DATE	LOCA-TION	PAST	ASCENTS IMMEDIATELY PRECEDING	ACCI-DENT	WHERE & WHEN DISCOVERED	SIGNS AND SYMPTOMS	HOW SOON TREATED	RX TABLES	SEQUELAE, ETC.	REMARKS	MI
61	B	B., G	4/2/57	N.L.		Limb pains after treating embolism case at 220 ft. for 38 minutes, water work previously.		8 hours		11 hours	Table 1			23
62	S	D., J.P.	5/24/57	P.H.		50# chbr (good) 18 ft. F.A.	50 ft. F.A.	At surface	Substernal pain		No recom-pression		X-ray showed pulmonary histoplasmosis positive histoplasmin skin test. Not disqualified from S/M	31
						SUBMARINE ESCAPE TRAINING CASUALITIES - RECOMPRESSION CHAMBER TESTS								
63	S	H., G.C.	3/22/44	N.L.			50# pr.	10 ft. on ascent	Subcutaneous and mediastinal emphysema, hoarse cyanosis		No recom-pression			8
64	A	F., R.L.	11/29/44	N.L.	Unsucessful 50# per week previously		50# pr	Few minutes after surfac-ing	Unconsciousness, paralysis, con-vulsions, visual disturbances, vomiting		165 ft. (73.4#)		Held breath on ascent	32
65	A(7B)	S., R.E.	5/10/46	P.H.			50# pr. (for 7 min)	At 10 ft. on ascent	Lt. arm and flank pain, visual dis-turbances, weakness, rash	30 min.	Table 3			33
66	M	C., R.N.	11/12/46	P.H.			50# pr. (for 9 min.)	On bottom	Subjective numbness, paralysis, etc., hysterical and dizzy on descent	22 min.	Table 3			34
67	M	B., J.G.	1/20/47	N.L.			99 ft. (74 min)	? chbr test	Right arm warmer than left		Table 1			?
68	M	O., H., Jr.	1/4/50	N.L.			50# pr.		Visual disturb-ances, rash-? allergy		No. RX		Subsequent pressure and ascents without difficulty	35
69	B	S., R.L.A.	9/15/50	N.L.	Instructor		165 ft. (73.4#)	1 hour after surfacing	Skin and limb bends, visual disturbances	1-1/2 hours	Table 3 and 2		Chamber run evening after water work (treating sus-pected casualty)	19

• SEE M.D.'S CODE ON LAST PAGE.

NO.	CASU-ALTY	NAME	DATE	LOCA-TION	PAST	ASCENTS IMMEDIATELY PRECEDING	ACCI-DENT	WHERE & WHEN DISCOVERED	SIGNS AND SYMPTOMS	HOW SOON TREATED	RX TABLES	SEQUELAE, ETC.	REMARKS	MD*
70	AS	H., J.T.	5/26/51	P.H.			50# pr.	At 10 ft. on ascent, and 5 minutes after surfacing	Dizzy, visual disturbances, voice loss, vertigo, chest fullness	13 min.	Table 3			21
71	A	M., G.D.	10/22/52	P.H.			50# pr.	20 ft. on ascent	Dizziness, fall to right, tinnitus, deafness on right		Table 3			?
72	A	B., R.F.	10/23/52	N.L.			50# pr.	Upon surfacing	Unconsciousness, paralysis, convulsions	1/2 hr	Table 4			36 et al
73	A	D., O.K.	2/8/54	N.L.			50# pr.	At surface	Convulsions, eyeballs fixed	1 minute	Table 3			37
74	M	F., J.P.	5/6/54	N.L.			50# pr.	At 25 ft. on ascent	Hysteria -- epistaxis with dizziness, anesthesia, nausea	3 minutes	Table 3 abbreviated			37
75	S	M., J.L.	7/7/57	N.L.			50# pr.	Later in day	Ear, neck, and throat pain, subcutaneous emphysema, hoarseness, dysphagia, mediastinal emphysema	10 hours	Table 3			24
76	A	H., J.D.	10/20/54	British			50# pr.		Convulsions, unconsciousness	?	Table 3 Recurrance at 40 ft., and recompression on Table 4			38 & 41
77	A(TB)	P., E.E.	9/30/54	Orion			90 ft. pr.		Anesthesia, knee pain		Table 3	Convulsions & unconsciousness after RX. Recompressed on tables 3 and 4		39 & 19
78	A	S., P.P.	1/20/56	N.L.			50# pr.	2 min. after surfacing	Anesthesia, paresthesia, confusion, paresis	3 minutes	Table 3		? aeroembolism or "fast tissue bends"	4C
79	AS	M., J.E.	9/25/56	N.L.			50# pr.	Few minutes after surfacing	paraesthesia, vertigo, headache, nausea, increased pressure in neck	10 minutes	Table 3			?

*SEE M.D.'S CODE ON LAST PAGE.

Medical Officers Code Numbers

1 -- BROWN	23 -- BOND
2 -- MAC CLATCHIE	24 -- SCHULTE
3 -- ADAMS	25 -- KIRKER
4 -- BEHNKE	26 -- STARK
5 -- JOHNSON	27 -- BARTON
6 -- CHRISMAN	28 -- SEIDEL
7 -- WILLMON	29 -- AKIN
8 -- YOUNG	30 -- MOSES
9 -- DUFFNER	31 -- HOLLER
10 -- COLE	32 -- CHRISMAN
11 -- HAYTER	33 -- GRATTON
12 -- KOPECKY	34 -- RITCHIE
13 -- KINSEY	35 -- GRANT
14 -- EDWARDS	36 -- FAUCETT
15 -- NEWMAN	37 -- BORUM
16 -- ICE	38 -- RUGS
17 -- DYKHUIZEN	39 -- JAMES
18 -- REILLY	40 -- GILLEN
19 -- EBERSOLE	41 -- GUNN
20 -- ALVIS	42 -- DOBBINS
21 -- MEANS	43 -- LAWSON
22 -- WAITE	44 -- BERNARD